What’s The Diagnosis of That Patient With Cough, Wheeze and/or Shortness of Breath

A recent article from the American Journal of Respiratory and Critical Care Medicine took major exception to the use of the terms: “reactive airways” and “reactive airways disease” (1). The authors stated that the use of these diagnostic labels “at best is an annoyance to those of us who want to maintain diagnostic clarity . . .” and “at worst, the terms represent a form of diagnostic laziness that may cause harm to patients”. One should note that the code book used by medical records personnel does not assign a diagnostic code to “reactive airways” or “reactive airways disease” but rather indicates the terms should be assigned the diagnostic code for asthma.

For the purpose of diagnosing work-related asthma, with all its potential economic and social consequences, diagnostic clarity is essential. Not to overstate the obvious it is impossible for a patient to have work-related asthma if the patient does not have asthma. All too often “reactive airways” or “reactive airways disease” are used to label patients with repeated respiratory symptoms without diagnostic tests to confirm asthma.

 Patients with asthma have respiratory symptoms and objective evidence of “airway hyperreactivity”. The latter is documented by a significant response to a bronchodilator, variability in peak flow testing or a significant bronchoconstrictor response to stimuli such as methacholine or cold air. “Airway hyperreactivity” has been described in other diseases besides asthma and it is a physiological abnormality, not a diagnosis.

“Reactive Airways Dysfunction Syndrome” (RADS), on the other hand, is a diagnosis. It is a type of work-related asthma where disease occurs after an exposure to a high concentration of an irritating substance. Specific criteria for “RADS” include evidence of “airway hyperreactivity” (Table I).

The use of the term “chronic obstructive pulmonary disease” (COPD) has also varied among clinicians. A consensus committee of the American Thoracic Society (ATS) defined COPD “as a disease state characterized by the presence of airflow obstruction due to chronic bronchitis or emphysema”. However, unremitting asthma with airway remodeling was included under the rubric of COPD (2). A recent consensus workshop from the National Heart Lung and Blood Institute and the World Health Organization has redefined COPD as “a disease state characterized by airflow limitation that is not fully reversible”. It goes on to state that COPD is “a mixture of small airway disease (obstructive bronchitis) and parenchymal destruction (emphysema), the relative contributions of which vary from person to person” (3).

In this latest publication, chronic asthma is no longer included under the rubric of COPD. Figures 1 and 2 illustrate the distinctions made between COPD and asthma although the document acknowledged that longstanding asthma on its own can lead to airway remodeling and partly irreversible airflow limitation. They conclude: “In
some patients with chronic asthma, a clear distinction from COPD is not possible using current imaging and physiological testing techniques, and it is assumed that asthma and COPD coexist in these patients” (3).

Particularly when the patient has a history of cigarette smoking, determination of whether work was a significant contributor to the development of the condition is facilitated by first reaching a decision on whether asthma or COPD is the predominant disease impairing the patient’s function. Although there is some overlap, the work exposures associated with the development of COPD (4,5) differ from those causing asthma (6). Once the diagnosis of the patient is determined, their previous exposures can be reviewed to reach a conclusion on the importance of work exposures in the etiology of their symptoms/impairment.

Diagnostic accuracy is useful for more than generating a semantic argument. Accuracy in diagnosis is important not only in determining the proper treatment modalities but also in developing a logical approach to determining the importance of various exposures to the etiology of the patient’s symptoms. Diagnostic accuracy increases the likelihood that medical advice provided about medical restrictions or leaving work is correct. This is extremely important given the social and economic costs of such medical opinions and increases the likelihood that such medical opinions will withstand the scrutiny of skeptical employers and an adversarial legal system.

<table>
<thead>
<tr>
<th>Table I. Cardinal Diagnostic Features of RADS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Identification of date, time(s), frequency and extent of exposure; the latter may be a single high exposure, multiple high exposures, or multiple somewhat less high exposures (yet still higher than either TLV or PEL concentrations)</td>
</tr>
<tr>
<td>Symptoms appear within 24 hr</td>
</tr>
<tr>
<td>No latency period between exposure and symptoms</td>
</tr>
<tr>
<td>Symptoms less likely to improve away from work</td>
</tr>
<tr>
<td>Objective (pulmonary function) tests demonstrate obstruction</td>
</tr>
<tr>
<td>Presence and persistence of nonspecific bronchial hyperresponsiveness (as measured by methacholine or histamine challenge tests)</td>
</tr>
</tbody>
</table>
Figure 1. Asthma and COPD

Asthma
Sensitizing agent

COPD
Noxious agent

Asthmatic airway inflammation
CD4+ T lymphocytes
Eosinophils

COPD airway inflammation
CD6+ T lymphocytes
Macrophages Neurophils

Completely reversible
Airflow Imitation
Completely irreversible

Figure 2. Characteristics of Inflammation in COPD and Asthma

<table>
<thead>
<tr>
<th>COPD</th>
<th>Asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cells</strong></td>
<td></td>
</tr>
<tr>
<td>Neutrophils</td>
<td>Eosinophils</td>
</tr>
<tr>
<td>Large increase in macrophages</td>
<td>Small increase in macrophages</td>
</tr>
<tr>
<td>Increase in CD8+ T lymphocytes</td>
<td>Increase in CD4+ Th2 lymphocytes</td>
</tr>
<tr>
<td>Activation of mast cells</td>
<td></td>
</tr>
<tr>
<td><strong>Mediators</strong></td>
<td></td>
</tr>
<tr>
<td>LTB4</td>
<td>LTD4</td>
</tr>
<tr>
<td>IL-8</td>
<td>IL-4, IL-5</td>
</tr>
<tr>
<td>TNF-α</td>
<td>(Plus many others)</td>
</tr>
<tr>
<td><strong>Consequences</strong></td>
<td></td>
</tr>
<tr>
<td>Squamous metaplasia of epithelium</td>
<td>Fragile epithelium</td>
</tr>
<tr>
<td>Parenchymal destruction</td>
<td>Thickening of basement membrane</td>
</tr>
<tr>
<td>Mucus metaplasia</td>
<td>Mucus metaplasia</td>
</tr>
<tr>
<td>Glandular enlargement</td>
<td>Glandular enlargement</td>
</tr>
<tr>
<td><strong>Response to Treatment</strong></td>
<td></td>
</tr>
<tr>
<td>Glucocorticosteroids have little or no effect</td>
<td>Glucocorticosteroids inhibit inflammation</td>
</tr>
</tbody>
</table>

References

3. www.goldcopd.com/workshop
6. www.asmanet.com/asthmapro/asmawork.htm#start
In this issue:
  What’s the Diagnosis of that Patient...
Advisory Board

John J. Bernick, M.D., Ph.D.
Representative, Michigan Occupational Medical Association
Raymond Demers, M.D., M.P.H.
Henry Ford Hospital
Michael Harbut, M.D., M.P.H.
Center for Occupational and AFL-CIO, Medical Advisor
Dana G. Kissner, M.D.
President, Michigan Thoracic Society
Steven Kreshover, M.D.
President, Michigan Allergy and Asthma Society
Thomas G. Robins, M.D., M.P.H.
University of Michigan
School of Public Health
Division of Occupational Medicine

Project SENSOR Staff

At the Michigan Department of Consumer and Industry Services

Douglas J. Kalinowski, C.I.H., M.S., Deputy Director
Bureau of Safety and Regulations
Project SENSOR, Co-Director
Bill Deliefde, M.P.H.
Regional Supervisor
Project SENSOR-MDCIS Liaison
John Peck, C.I.H., M.S., Chief
Occupational Health Division
Debbie Wood
Division Chief Secretary

At Michigan State University—College of Human Medicine

Kenneth D. Rosenman, M.D.
Professor of Medicine
Project SENSOR, Co-Director
Mary Jo Reilly, M.S.
Project SENSOR Coordinator
Amy Sims, B.S.
Project SENSOR NIHL Coordinator
Project SENSOR Office Staff:
Tracy Carey
Ruth VanderWaals
Patient Interviewers:
Beth Hanna, R.N.
Amy Krizek

Michigan Law Requires the Reporting of Known or Suspected Occupational Diseases

Reporting can be done by:

FAX
(517) 432-3606
Telephone
1-800-446-7805
E-Mail
ODREPORT@ht.msu.edu
Web
www.chm.msu.edu/oem
Mail
Michigan Department of Consumer and Industry Services
Division of Occupational Health
P.O. Box 30649
Lansing, MI 48909-8149

Reporting forms can be obtained by calling (517) 322-5208
Or
1-800-446-7805

The Project SENSOR News is published quarterly by Michigan State University-College of Human Medicine with funding from the Michigan Department of Consumer and Industry Services and is available at no cost. Suggestions and comments are welcome.

(517) 353-1846
MSU-CHM
117 West Fee Hall
East Lansing, MI 48824-1316